(two), fatigue (two), insomnia (one), back pain (two).

In one omeprazole treated patient the serum aspartate aminotransferase activity rose from 20 to 82 U/l (reference range 20-40 U/l) and the alkaline phosphatase activity from 368 to 808 U/l (reference range 82-224 U/l). The patient had no symptoms and the values were almost normal one month after the end of treatment. Two cimetidine treated patients also had raised aspartate aminotransferase activities. Some patients in both groups had abnormal liver function values at entry, which continued unchanged during the trial. In several patients single laboratory values fell outside the normal range, but these abnormalities occurred at random in both groups. The mean change in laboratory values in the two treatment groups from day 1 to day 43 was compared by using the two sample *t* test. No significant differences were found ($p>0\cdot15$), apart from the difference caused by a slight increase in the serum creatinine concentration in the cimetidine group ($p=0\cdot00004$).

Discussion

Previous controlled studies have provided little information on the effect of omeprazole on the healing of ulcers of the body of the stomach. A German multicentre trial in patients with gastric ulcer found no appreciable difference between the effects of omeprazole 20 mg and ranitidine 300 mg daily.[6] The ulcer was located in the body of the stomach in 53 patients, and the authors mentioned that the healing rate in this subgroup was lower than among the remaining patients, regardless of treatment. In two other trials in patients with gastric ulcer omeprazole was found to be superior to ranitidine but the available abstracts provide no information on sites of the ulcers.[7,8] In one study, which comprised solely patients with prepyloric ulcers, treatment with omeprazole 30 mg was found to be slightly superior to treatment with cimetidine 1 g.[3]

We conclude that omeprazole 30 mg daily accelerates the healing of ulcers of the body of the stomach as compared with cimetidine 1 g daily. The results suggest that this effect may be more pronounced in larger ulcers than in smaller ulcers. A trial in patients with mixed gastric ulcers showed that omeprazole 40 mg daily provides higher healing rates than omeprazole 20 mg daily.[7] Possibly, therefore, our results could be improved by using a higher dose.

Our findings also support the conclusion from studies of duodenal ulcers, prepyloric ulcers, and mixed gastric ulcers that short term treatment of peptic ulcers with omeprazole is safe. The safety and efficacy of longer term use of omeprazole have yet to be assessed.

Omeprazole and placebo capsules were kindly provided by A B Hässle, Mölndal, Sweden. Cimetidine and placebo were kindly provided by Smith Kline and French Ltd, Welwyn Garden City, England. We thank Mr Kjeld Clemmensen-Rotne, Astra-Gruppen A/S, Albertslund, Denmark, for skilfully monitoring the conduct of the study.

Collaborating centres and participating clinicians—Department of Medical Gastroenterology, Odense University Hospital: K Lauritsen, L S Laursen, T Havelund, P Bytzer, J Rask-Madsen; Departments of Medical and Surgical Gastroenterology, Frederiksberg Hospital: Dorthe Teilum, J Eriksen, H D Petersen, Karen M Olesen; Department of Surgery, Horsens Hospital: J Ørnsholt; Department of Medicine F, Gentofte Hospital: E Gudmand-Høyer, Hanne S Jensen, J J Rumessen, L Ovesen, Anne G Mikkelsen; Department of Surgery K, Vejle Hospital: C K Axelsson; Department of Surgical Gastroenterology, Rigshospitalet, Copenhagen: Linda Bardram, S Boesby; Department of Surgical Gastroenterology F, Bispebjerg Hospital, Copenhagen: A Hjortrup, J G Stage, O G Backer; Department of Surgery, Ribe Hospital: J H Olsen; Department of Medical Gastroenterology, Herlev Hospital: P Schlichting, M Vilien, H R Wulff; Department of Medical Gastroenterology, Glostrup Hospital: S J Rune, Hanne Skovbjerg; Department of Medicine, Central Hospital, Hillerød: L Elsborg, P J Ranløv.

1 Walt RP, Gomes M de FA, Word EC, Logan KH, Pounder RE. Effect of daily oral omeprazole on 24 hour intragastric acidity. *Br Med J* 1983;**287**:12-4.
2 Lauritsen K, Rune SJ, Bytzer P, *et al*. Effect of omeprazole and cimetidine on duodenal ulcer: a double blind comparative trial. *N Engl J Med* 1985;**312**: 958-61.
3 Lauritsen K, Rune SJ, Wulff HR, *et al*. Effect of omeprazole and cimetidine on prepyloric gastric ulcer. Double blind comparative trial. *Gut* 1988;**29**:249-53.
4 Brooks FP. The pathophysiology of peptic ulcer disease. *Dig Dis Sci* 1985; **30**(suppl):155-295.
5 Wulff HR, Schlichting P. *MEDSTAT. Statistical program for the analysis of controlled therapeutic trials. Version 2*. Copenhagen: Astra, 1985.
6 Classen M, Dammann HG, Domschke W, *et al*. Abheilungsraten nach Omeprazol- und Ranitidin-Behandlung des Ulcus ventriculi. Ergebnisse einer deutschen Multizenterstudie. *Dtsch Med Wochenschr* 1985;**110**:628-33.
7 Walan A, Bader JP, Classen M, *et al*. Omeprazole and ranitidine in the treatment of benign gastric ulcer. An international multicentre study [Abstract]. *J Gastroenterol* 1987;**25**:582.
8 Barbara L, Saggioro A, Olsson J, Cisternino M, Franceschi M. Omeprazole 20 mg and ranitidine 150 mg bd in the healing of benign gastric ulcers. An Italian multicentre study [Abstract]. *Gut* 1987;**28**:A1341.

(Accepted 10 January 1989)

Hypothyroidism in polymyalgia rheumatica and giant cell arteritis

P Wiseman, K Stewart, G S Rai

Department of Geriatric Medicine, Whittington Hospital, London N19 5NF
P Wiseman, MRCP, *senior registrar*
K Stewart, MRCP, *senior registrar*
G S Rai, FRCP, *consultant physician and senior lecturer*

Correspondence to:
Dr Rai.

Br Med J 1989;**298**:647-8

In 1977 a single case report by How *et al* highlighted the possible coexistence of giant cell arteritis and hypothyroidism and suggested a common cause.[1] We set up a study to assess whether this association between hypothyroidism and giant cell arteritis, and also polymyalgia rheumatica, existed and whether it might have an immunological basis.

Patients, methods, and results

The notes of all patients treated for polymyalgia rheumatica and giant cell arteritis in the department of geriatric medicine at this and the Royal Northern Hospitals during 1981-7 were retrieved and the patients' thyroid state noted. The two conditions had been diagnosed from clinical features, the erythrocyte sedimentation rate, temporal artery biopsy when indicated, and the response to steroids. In patients in whom temporal artery biopsy had not been done the diagnosis was accepted only if steroids had produced the expected clinical response.

Hypothyroid patients who had not been screened for thyroid antibodies were screened prospectively, but only 11 of the 15 patients could be traced. Thyroid function tests were also repeated in these patients. Patients with hypothyroidism were grouped according to whether it had developed before or at the time of presentation or during follow up and were compared with the euthyroid group for age, sex, erythrocyte sedimentation rate, haemoglobin concentration, and platelet count.

We identified 36 patients (26 women and 10 men; mean age 80·5 years) with giant cell arteritis (20 patients) or polymyalgia rheumatica (16) (table). Temporal artery biopsy was not done in 11 of the patients with giant cell arteritis as the diagnosis was clear. Thyroid function tests showed that 15 of the 31

Time of onset of hypothyroidism related to presentation with polymyalgia rheumatica or giant cell arteritis

	No of patients	No with hypothyroidism	Time of onset of hypothyroidism: Before or at presentation	Time of onset of hypothyroidism: After presentation
Giant cell arteritis	20	6	3	3
Polymyalgia rheumatica	16	9	7	2

patients tested were hypothyroid. The table shows the time of onset of the hypothyroidism; in five patients it developed during follow up, after six months to 10 years.

There was no significant difference in mean age, erythrocyte sedimentation rate, haemoglobin concentration, or platelet count between the hypothyroid and euthyroid patients. As expected, there were proportionately more women than men (ratio 12:3) among those with hypothyroidism. Thyroid autoantibodies were present in seven of the 11 patients screened prospectively (five with giant cell arteritis and two with polymyalgia rheumatica).

Comment

Hypothyroidism occurred concurrently with polymyalgia rheumatica and giant cell arteritis or developed during follow up in a high proportion of patients (15/36). This has important implications for the treatment of patients in whom the rheumatic symptoms of hypothyroidism[2] could be misconstrued as an exacerbation of their symptoms, resulting in unnecessary increases in the steroid dose. This happened in one of our patients: the typical symptoms of polymyalgia rheumatica responded to steroid treatment, but a year later she developed aches and pains with general malaise suggesting a relapse of the disease. At this stage her erythrocyte sedimentation rate was normal, although her mean corpuscular volume was raised. Her steroid dose was increased, but her condition did not improve. Measurement of the serum thyroxine and thyroid stimulating hormone concentrations confirmed the development of primary hypothyroidism. Her symptoms improved with thyroxine replacement. Despite remaining euthyroid she had one relapse of her polymyalgia rheumatica when her steroid dose was reduced.

The association of the two conditions supports the idea of a common autoimmune aetiology. More work is needed to assess the importance, if any, of thyroid autoantibodies in euthyroid patients with polymyalgia rheumatica or giant cell arteritis.

1 How J, Bewsher PD, Walker V. Giant cell arteritis and hypothyroidism. *Br Med J* 1977;ii:99.
2 Golding DN. The musculo-skeletal features of hypothyroidism. *Postgrad Med J* 1971;**47**:611-4.

(Accepted 8 December 1988)

Revision arthroplasty: a high price to pay

Clark R Dreghorn, David L Hamblen

Department of Orthopaedic Surgery, Western Infirmary, Glasgow G11 6NT
Clark R Dreghorn, FRCS, *orthopaedic senior registrar*
David L Hamblen, FRCS, *professor of orthopaedic surgery*

Correspondence to:
Mr C R Dreghorn, University Department of Orthopaedic Surgery, Clinical Sciences Building, St James's University Hospital, Leeds LS9 7TF.

Br Med J 1989;**298**:648-9

Expenditure in the NHS has never been under greater scrutiny. Although total hip replacement is one of the most cost effective advances in medical technology,[1] many orthopaedic surgeons are facing criticisms about its costs and are under pressure to reduce unit costs. In 1983 we assessed the costs of primary joint replacements.[2] We have now evaluated the costs of all revision arthroplasties carried out in the same year to make direct comparisons.

Methods and results

The time to failure of the replacement joint and any investigations performed to determine the cause of failure were noted from the patients' case records. The duration of inpatient stay was calculated, including the time spent in hospital for treatment of complications as a result of failure of the primary arthroplasty in addition to the perioperative period itself. Details of the operative procedure and any antibiotics prescribed were noted. Prices current when the expenditure was incurred were obtained from the hospital's pharmacy and stores department records.

The table gives data on all revision hip and knee arthroplasties performed in 1983. The interval from primary to revision surgery showed no notable difference between patients with and without infection of the joint, but those with infection had a longer mean duration of inpatient stay (hip replacement 73 *v* 28 days; knee replacement 47 *v* 42 days).

Eight of the 27 patients who had revision hip replacements and four of the 13 who had revision knee replacements required admission before their revision surgery, spending on average 30 and 69 additional days in hospital respectively.

There is a fixed policy for antibiotic prophylaxis in primary joint replacement, and bone cement containing antibiotic was not used in any primary operations. This allowed a direct comparison to be made between the costs of antibiotics at primary and revision arthroplasty (£8.69 *v* £98).

The greater range of implants used in revision arthroplasty resulted in a higher average cost for each revision operation compared with the equivalent primary procedure (hips £119 *v* £108, knees £442 *v* £361).

Details of revision joint replacement in 1983. Figures are numbers of patients except where stated otherwise

	Hip	Knee
Primary arthroplasties	216	61
Revision arthroplasties	27	13
Mean (range) interval to revision (years)	6·3 (1·8-13·0)	5 (1·8-10·3)
Cause of revision procedure:		
Mechanical loosening	18	7
Infection	6	5
Other	3	1
Duration of perioperative hospital stay (days):		
Primary arthroplasty	20	31
Revision arthroplasty	38	43
Type of revision:		
Exchange arthroplasty	24	12
Excision arthroplasty	3	1
Antibiotic use during revision procedure:		
Systemic antibiotic used	26	13
Duration of treatment (days)	18	28
% Increase in duration of treatment over primary arthroplasty	419	393
Antibiotic cement used	16/24	9/12
Total cost/patient (£)	90	114

Comment

Since 1970 the number of revision arthroplasties being performed each year at this hospital has increased progressively from 10 a year in 1970-4 to 22 a year in 1981-5. Our previous study showed that almost 90% of the expenditure on a joint replacement (£2440 out of £2730) was due to the hotel costs incurred. These new data show that costs are greatly increased when patients require revision operations, not only at the